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 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  FEB 25   CA/CAPLUS - Russian Agency for Patents and Trademarks
                   (ROSPATENT) added to list of core patent offices covered
 NEWS  4  FEB 28   PATDPAFULL - New display fields provide for legal status
                   data from INPADOC
 NEWS  5  FEB 28   BABS - Current-awareness alerts (SDIs) available
 NEWS  6  FEB 28   MEDLINE/LMEDLINE reloaded
 NEWS  7  MAR 02   GBFULL: New full-text patent database on STN
 NEWS  8  MAR 03   REGISTRY/ZREGISTRY - Sequence annotations enhanced
 NEWS  9  MAR 03   MEDLINE file segment of TOXCENTER reloaded
 NEWS  10 MAR 22   KOREAPAT now updated monthly; patent information enhanced
 NEWS  11 MAR 22   Original IDE display format returns to REGISTRY/ZREGISTRY
 NEWS  12 MAR 22   PATDPASPC - New patent database available
 NEWS  13 MAR 22   REGISTRY/ZREGISTRY enhanced with experimental property tags
 NEWS  14 APR 04   EPFULL enhanced with additional patent information and new
                   fields
 NEWS  15 APR 04   EMBASE - Database reloaded and enhanced
 NEWS  16 APR 18   New CAS Information Use Policies available online
 NEWS  17 APR 25   Patent searching, including current-awareness alerts (SDIs),
                   based on application date in CA/CAplus and USPATFULL/USPAT2
                   may be affected by a change in filing date for U.S.
                   applications.
 NEWS  18 APR 28   Improved searching of U.S. Patent Classifications for
                   U.S. patent records in CA/CAplus

 NEWS EXPRESS  JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE 'HOME' ENTERED AT 14:23:43 ON 09 MAY 2005

=> index bioscience
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
       AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,
       BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
       CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 14:23:54 ON 09 MAY 2005

75 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
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=> s preconditioning
        370   FILE ADISCTI
         18   FILE ADISINSIGHT
         14   FILE ADISNEWS
        275   FILE AGRICOLA
         70   FILE ANABSTR
         14   FILE ANTE
         27   FILE AQUALINE
        140   FILE AQUASCI
        121   FILE BIOBUSINESS
          4   FILE BIOCOMMERCE
        131   FILE BIOENG
       6800   FILE BIOSIS
        109   FILE BIOTECHABS
        109   FILE BIOTECHDS
        412   FILE BIOTECHNO
        657   FILE CABA
        315   FILE CANCERLIT
       5254   FILE CAPLUS
         54   FILE CEABA-VTB
         15   FILE CIN
        267   FILE CONFSCI
         15   FILE CROPB
         59   FILE CROPU
          5   FILE DDFB
        869   FILE DDFU
         76   FILE DGENE
        553   FILE DISSABS
          5   FILE DRUGB
        994   FILE DRUGU
         82   FILE EMBAL
       4241   FILE EMBASE
       2600   FILE ESBIOBASE
        206   FILE FEDRIP
         65   FILE FROSTI
         98   FILE FSTA
         61   FILE GENBANK
          9   FILE HEALSAFE
       1201   FILE IFIPAT
          3   FILE IMSDRUGNEWS
          4   FILE IMSRESEARCH
        711   FILE JICST-EPLUS
          7   FILE KOSMET
        526   FILE LIFESCI
          6   FILE MEDICONF
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      4881   FILE MEDLINE
        24   FILE NIOSHTIC
       583   FILE NTIS
  52 FILES SEARCHED...
        75   FILE OCEAN
      5144   FILE PASCAL
         2   FILE PHAR
         3   FILE PHARMAML
        18   FILE PHIN
       306   FILE PROMT
         6   FILE PROUSDDR
         9   FILE RDISCLOSURE
      8243   FILE SCISEARCH
      2045   FILE TOXCENTER
      4450   FILE USPATFULL
       361   FILE USPAT2
         8   FILE VETB
        14   FILE VETU
        99   FILE WATER
       718   FILE WPIDS
         3   FILE WPIFV
       718   FILE WPINDEX

  65 FILES HAVE ONE OR MORE ANSWERS,   75 FILES SEARCHED IN STNINDEX

L1   QUE PRECONDITIONING

=> d rank
F1        8243   SCISEARCH
F2        6800   BIOSIS
F3        5254   CAPLUS
F4        5144   PASCAL
F5        4881   MEDLINE
F6        4450   USPATFULL
F7        4241   EMBASE
F8        2600   ESBIOBASE
F9        2045   TOXCENTER
F10       1201   IFIPAT
F11        994   DRUGU
F12        869   DDFU
F13        718   WPIDS
F14        718   WPINDEX
F15        711   JICST-EPLUS
F16        657   CABA
F17        583   NTIS
F18        553   DISSABS
F19        526   LIFESCI
F20        412   BIOTECHNO
F21        370   ADISCTI
F22        361   USPAT2
F23        315   CANCERLIT
F24        306   PROMT
F25        275   AGRICOLA
F26        267   CONFSCI
F27        206   FEDRIP
F28        140   AQUASCI
F29        131   BIOENG
F30        121   BIOBUSINESS
F31        109   BIOTECHABS
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F38          70   ANABSTR
F39          65   FROSTI
F40          61   GENBANK
F41          59   CROPU
F42          54   CEABA-VTB
F43          27   AQUALINE
F44          24   NIOSHTIC
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F46          18   PHIN
F47          15   CIN
F48          15   CROPB
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F50          14   ANTE
F51          14   VETU
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F53           9   RDISCLOSURE
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F56           6   MEDICONF
F57           6   PROUSDDR
F58           5   DDFB
F59           5   DRUGB
F60           4   BIOCOMMERCE
F61           4   IMSRESEARCH
F62           3   IMSDRUGNEWS
F63           3   PHARMAML
F64           3   WPIFV
F65           2   PHAR


=> file f1, f2, f3, f4, f5, f6, f7, f8, f9
COST IN U.S. DOLLARS                        SINCE FILE      TOTAL
                                               ENTRY    SESSION
FULL ESTIMATED COST                             1.18       1.39

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FILE 'ESBIOBASE' ENTERED AT 14:25:03 ON 09 MAY 2005
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FILE 'TOXCENTER' ENTERED AT 14:25:03 ON 09 MAY 2005

=> s preconditioning (p) mitochondria
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FIELD CODE - 'AND' OPERATOR ASSUMED 'DITIONING (P) MITOCHOND'
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'DITIONING (P) MITOCHOND'
L2 1261 PRECONDITIONING (P) MITOCHONDRIA

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3 508 DUP REM L2 (753 DUPLICATES REMOVED)

=> s L3 AND (isocitrate(w)dehydrogenase(w)NAD(w)alpha(w)subunit OR succinyl(w)CoA(w)ligase OR ATP(w)synthase OR dihyrolipoamide(w)succinyltransferase OR ubiquinol(w)cytochrome(w)c(w)oxidoreductase OR NADH(w)ubiquinone(w)oxidoreductase)
6 FILES SEARCHED...
L4 15 L3 AND (ISOCITRATE(W) DEHYDROGENASE(W) NAD(W) ALPHA(W) SUBUNIT OR SUCCINYL(W) COA(W) LIGASE OR ATP(W) SYNTHASE OR DIHYROLIPOAMIDE(W) SUCCINYLTRANSFERASE OR UBIQUINOL(W) CYTOCHROME(W) C(W) OXIDOREDUCTASE OR NADH(W) UBIQUINONE(W) OXIDOREDUCTASE)

=> d l4 ibib ti abs 1-15

L4 ANSWER 1 OF 15 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:489036 SCISEARCH
THE GENUINE ARTICLE: 822TJ
TITLE: Diazoxide affects the IF1 inhibitor protein binding to F-1 sector of beef heart F(0)F(1)ATPsynthase
AUTHOR: Contessi S; Metelli G; Mavelli I; Lippe G (Reprint)
CORPORATE SOURCE: Univ Udine, Dept Biomed Sci & Technol, Ple Kolbe 4, I-33100 Udine, Italy (Reprint); Univ Udine, Dept Biomed Sci & Technol, I-33100 Udine, Italy; Univ Udine, MATI Ctr Excellence, I-33100 Udine, Italy
COUNTRY OF AUTHOR: Italy
SOURCE: BIOCHEMICAL PHARMACOLOGY, (15 MAY 2004) Vol. 67, No. 10, pp. 1843-1851.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.
ISSN: 0006-2952.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI Diazoxide affects the IF1 inhibitor protein binding to F-1 sector of beef heart F(0)F(1)ATPsynthase

AB Diazoxide, a selective opener of the mitochondrial ATP-sensitive K+ channel (mitoK(ATP)), has been reported to enhance F(0)F(1)**ATP-synthase** inhibition during ischemia, but the underlying mechanisms are still unclear. Here, we demonstrate that diazoxide directly interacts with the F-1 sector of beef heart F(0)F(1)ATPsynthase markedly promoting the binding of the inhibitor protein (IF1) to beta subunit. More specifically, the treatment of soluble F-1 with one equivalent of diazoxide was sufficient to decrease the K-d of IF1-F-1 complex at low pH. Such effect was revealed only on the cycling enzyme, while no effect was observed in the absence of Mg-ATP. However, diazoxide binding occurred independently from the catalysis, as shown by the structural changes induced by the drug in not catalytically active F-1 and revealed by CD

spectra. In addition, kinetic analysis of ATP hydrolysis demonstrated that diazoxide exerts a stabilising role on Mg-ADP bound in the catalytic site of the beta subunit adopting the tight conformation (beta(DP)). In accordance, a stabilising effect of Mg-ADP at the nucleotide binding domain (NBD) has been reported also for K-ATP channel. These results suggest that diazoxide binds to beta subunit at NBD, which is highly conserved in the ATP-binding cassette protein family, thus inducing nucleotide stabilisation and favouring F-1 conformation suitable for IF, binding. Finally, diazoxide also increased IF1 binding to membrane bound F1, while it did not influence the energisation-dependent IF1 release. As IF1 binding mediates the F(0)F(1)ATPsynthase inhibition, we suggest that such mechanism may contribute to cardioprotection during ischemia. (C) 2004 Elsevier Inc. All rights reserved.

L4 ANSWER 2 OF 15 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:972343 SCISEARCH
THE GENUINE ARTICLE: 739VH
TITLE: ATP-sensitive K+ channels in renal mitochondria
AUTHOR: Cancherini D V; Trabuco L G; Reboucas N A; Kowaltowski A J (Reprint)
CORPORATE SOURCE: Av Prof Lineu Prestes 748, Cidade Univ, BR-05508900 Sao Paulo, Brazil (Reprint); Univ Sao Paulo, Dept Fisiol & Biofis, Inst Ciencias Biomed, BR-05508900 Sao Paulo, Brazil; Univ Sao Paulo, Dept Bioquim, Inst Quim, BR-05508900 Sao Paulo, Brazil
COUNTRY OF AUTHOR: Brazil
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY-RENAL PHYSIOLOGY, (DEC 2003) Vol. 285, No. 6, pp. F1291-F1296.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 0363-6127.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 38

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI ATP-sensitive K+ channels in renal mitochondria

AB Isolated kidney mitochondria swell when incubated in hyposmotic solutions containing K+ salts in a manner inhibited by ATP, ADP, 5-hydroxydecanoate, and glibenclamide and stimulated by GTP and diazoxide. These results suggest the existence of ATP-sensitive K+ channels in these mitochondria, similar to those previously described in heart, liver, and brain. Renal mitochondrial ATP-sensitive K+ uptake rates are similar to140 nmol . min(-1) . mg protein(-1). This K+ transport results in a slight increase in respiration and decrease in the inner membrane potential. In addition, the activation of ATP-inhibited K+ uptake using diazoxide leads to a decrease of ATP hydrolysis through the reverse activity of the F0F1 **ATP synthase** when respiration is inhibited. In conclusion, we characterize an ATP-sensitive K+ transport pathway in kidney mitochondria that affects volume, respiration, and membrane potential and may have a role in the prevention of mitochondrial ATP hydrolysis.

L4 ANSWER 3 OF 15 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:931072 SCISEARCH
THE GENUINE ARTICLE: 379KX
TITLE: Myocardial ischemic preconditioning and mitochondrial F1F0-ATPase activity
AUTHOR: Bosetti F (Reprint); Yu G Y; Zucchi R; RoncaTestoni S; Solaini G
CORPORATE SOURCE: NIA, SECT BRAIN PHYSIOL & METAB, NIH BLDG 10, ROOM 6N202, 9000 ROCKVILLE PIKE, BETHESDA, MD 20892 (Reprint); SCUOLA

SUPER STUDI UNIV & PERFEZIONAMENTO S ANNA, PISA, ITALY; UNIV PISA, SEZ CHIM & BIOCHIM MED, DIPARTIMENTO SCI UOMO & AMBIENTE, I-56100 PISA, ITALY
COUNTRY OF AUTHOR: USA; ITALY
SOURCE: MOLECULAR AND CELLULAR BIOCHEMISTRY, (DEC 2000) Vol. 215, No. 1-2, pp. 31-37.
Publisher: KLUWER ACADEMIC PUBL, SPUIBOULEVARD 50, PO BOX 17, 3300 AA DORDRECHT, NETHERLANDS.
ISSN: 0300-8177.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 39

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

TI Myocardial ischemic preconditioning and mitochondrial F1F0-ATPase activity

AB A short period of ischemia followed by reperfusion (ischemic **preconditioning**) is known to trigger mechanisms that contribute to the prevention of ATP depletion. In ischemic conditions, most of the ATP hydrolysis can be attributed to mitochondrial F1F0-ATPase (**ATP synthase**). The purpose of the present study was to examine the effect of myocardial ischemic **preconditioning** on the kinetics of ATP hydrolysis by F1F0-ATPase. **Preconditioning** was accomplished by three 3-min periods of global ischemia separated by 3 min of reperfusion. Steady state ATP hydrolysis rates in both control and preconditioned **mitochondria** were not significantly different. This suggests that a large influence of the enzyme on the **preconditioning** mechanism may be excluded. However, the time required by the reaction to reach the steady state rate was increased in the preconditioned group before sustained ischemia, and it was even more enhanced in the first 5 min of reperfusion (101 +/- 3.0 sec in preconditioned vs. 83.4 +/- 4.4 sec in controls, $p < 0.05$). These results suggest that this transient increase in activation time may contribute to the cardioprotection by slowing the ATP depletion in the very critical early phase of post-ischemic reperfusion.

L4 ANSWER 4 OF 15 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2004:692747 CAPLUS
DOCUMENT NUMBER: 141:241156
TITLE: Ischemic preconditioning exaggerates cardiac damage in PKC-δ null mice
AUTHOR(S): Mayr, Manuel; Metzler, Bernhard; Chung, Yuen-Li; McGregor, Emma; Mayr, Ursula; Troy, Helen; Hu, Yanhua; Leitges, Michael; Pachinger, Otmar; Griffiths, John R.; Dunn, Michael J.; Xu, Qingbo
CORPORATE SOURCE: Department of Cardiac and Vascular Sciences, St. George's Hospital Medical School, London, SW17 0RE, UK
SOURCE: American Journal of Physiology (2004), 287(2, Pt. 2), H946-H956
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Ischemic preconditioning exaggerates cardiac damage in PKC-δ null mice

AB Ischemic preconditioning confers cardiac protection during subsequent ischemia-reperfusion, in which protein kinase C (PKC) is believed to play an essential role, but controversial data exist concerning the PKC-δ isoform. In an accompanying study, the authors described metabolic changes in PKC-δ knockout mice. The authors now explore their effect on early preconditioning. Both PKC-δ-/- and PKC-δ+/+ mice underwent three cycles of 5-min left descending artery occlusion/5-min reperfusion, followed by 30-min occlusion and 2-h reperfusion. Unexpectedly, preconditioning exaggerated

ischemia-reperfusion injury in PKC-δ-/- mice. Whereas ischemic preconditioning increased superoxide anion production in PKC-δ+/+ hearts, no increase in reactive oxygen species was observed in PKC-δ-/- hearts. Proteomic anal. of preconditioned PKC-δ+/+ hearts revealed profound changes in enzymes related to energy metabolism, e.g., NADH dehydrogenase and **ATP synthase**, with partial fragmentation of these mitochondrial enzymes and of the E2 component of the pyruvate dehydrogenase complex. Interestingly, fragmentation of mitochondrial enzymes was not observed in PKC-δ-/- hearts. High-resolution NMR anal. of cardiac metabolites demonstrated a similar rise of phosphocreatine in PKC-δ+/+ and PKC-δ-/- hearts, but the preconditioning-induced increase in phosphocholine, alanine, carnitine, and glycine was restricted to PKC-δ+/+ hearts, whereas lactate concns. were higher in PKC-δ-/- hearts. Taken together, the authors' results suggest that reactive oxygen species generated during ischemic preconditioning might alter mitochondrial metabolism by oxidizing key mitochondrial enzymes and that metabolic adaptation to preconditioning is impaired in PKC-δ-/- hearts.

REFERENCE COUNT: 51 THERE ARE 51 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:668326 CAPLUS
DOCUMENT NUMBER: 139:290251
TITLE: Ischaemic preconditioning and a mitochondrial KATP channel opener both produce cardioprotection accompanied by F1F0-ATPase inhibition in early ischaemia
AUTHOR(S): Ala-Rami, Antti; Ylitalo, Kari V.; Hassinen, Ilmo E.
CORPORATE SOURCE: Department of Medical Biochemistry and Molecular Biology, University of Oulu, Oulu, 90014, Finland
SOURCE: Basic Research in Cardiology (2003), 98(4), 250-258
CODEN: BRCAB7; ISSN: 0300-8428
PUBLISHER: Steinkopff Verlag
DOCUMENT TYPE: Journal
LANGUAGE: English

TI Ischaemic preconditioning and a mitochondrial KATP channel opener both produce cardioprotection accompanied by F1F0-ATPase inhibition in early ischaemia

AB Ischemic preconditioning gives powerful protection against prolonged ischemia affecting several intracellular regulatory and messenger pathways, although their mutual importance is far from established. Protective, preconditioning-like effects have been reported for KATP channel openers, and most of the evidence points to the mitochondrial KATP channels. We show here that the KATP channel opener diazoxide, which acts selectively on the mitochondrial channel, causes potentiation of ischemic inhibition of mitochondrial **ATP synthase** (F1F0-ATPase) along with cardioprotection. These effects are comparable with that of ischemic preconditioning. The administration of diazoxide did not affect the cellular energy state as monitored with 31P NMR. The actions of both diazoxide and ischemic preconditioning were prevented by 5-hydroxydecanoate, a specific inhibitor of the mitochondrial KATP channel. Thus mitochondrial KATP channel opening and ischemic preconditioning must share common mechanisms of action involving mitochondrial F1F0-ATPase, although involvement of the energy state in protection could not be proved.

REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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ACCESSION NUMBER: 2004-0590987 PASCAL
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TITLE (IN ENGLISH): F.sub.0f.sub.1 **ATP synthase** activity is differently modulated by coronary reactive hyperemia before and after ischemic **preconditioning** in the goat

AUTHOR: PENNA Claudia; PAGLIARO Pasquale; RASTALDO Raffaella; DI PANCRAZIO Francesca; LIPPE Giovanna; GATTULLO Donatella; MANCARDI Daniele; SAMAJA Michele; LOSANO Gianni; MAVELLI Irene

CORPORATE SOURCE: Sezione di Fisiologia, Dipartimento di Neuroscienze and Dipartimento di Scienze Cliniche e Biologiche, Universita di Torino, 10100 Turin, United States; Dipartimento di Scienze e Tecnologie Biomediche and Centro di Eccellenza Micro gravity, Aging, Training, and Immobility, Universita di Udine, 33100 Udine, Italy; Dipartimento di Medicina, Chirurgia, e Odontoiatria, Universita di Milano, 20100 Milan, Italy

SOURCE: American journal of physiology. Heart and circulatory physiology, (2004), 56(5), H2192-H2200, 48 refs. ISSN: 0363-6135 CODEN: AJPPDI

DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670D, 354000122491250390

TIEN F.sub.0f.sub.1 **ATP synthase** activity is differently modulated by coronary reactive hyperemia before and after ischemic **preconditioning** in the goat

AN 2004-0590987 PASCAL

AB The amplitude of coronary reactive hyperemia (CRH), elicited by 15 s of ischemia, is reduced in hearts subjected to 5 min of ischemic **preconditioning** (IP). F.sub.0F.sub.1ATP synthase activity and ATP concentration are also altered by IP. We hypothesized that F.sub.0F.sub.1 **ATP synthase** is differently modulated by the inhibitor protein IF.sub.1 during CRH elicited before (CRH.sub.n.sub.p) and after (CRH.sub.p.sub.r.sub.e.sub.c) IP. Hemodynamic parameters were recorded in 10 anesthetized goats. Myocardial biopsies were obtained before IP (C.sub.n.sub.p), during CRH.sub.n.sub.p, 4 and 6 min after the onset of CRH.sub.n.sub.p, after IP (C.sub.p.sub.r.sub.e.sub.c), during CRH.sub.p.sub.r.sub.e.sub.c, and 4 min after CRH.sub.p.sub.r.sub.e.sub.c. F.sub.0F.sub.1ATP synthase activity, ATP concentration, and ATP-to-ADP ratio (ATP/ADP) were determined. Compared with CRH.sub.n.sub.p, IP blunted CRH.sub.p.sub.r.sub.e.sub.c. F.sub.0F.sub.1 **ATP synthase** activity transiently increased during CRH.sub.n.sub.p, decreased 4 min after CRH.sub.n.sub.p, and returned to control 2 min later; it was lower after IP (C.sub.p.sub.r.sub.e.sub.c) and did not change during and after CRH.sub.p.sub.r.sub.e.sub.c. All these changes in activity were modulated by IF.sub.1. During CRH.sub.n.sub.p, ATP concentration and ATP/ADP were reduced compared with C.sub.n.sub.p and began to rise 6 min thereafter. During C.sub.p.sub.r.sub.e.sub.c, both parameters were transiently reduced but increased during and after CRH.sub.p.sub.r.sub.e.sub.c. Hence, during CRH.sub.n.sub.p, F.sub.0f.sub.1 **ATP synthase** activity transiently increases and then decreases significantly. The shortlasting inhibition of the enzyme may explain why a few seconds of occlusion do not induce IP. After IP, F.sub.0F.sub.1 **ATP synthase** activity is blunted, and it is not affected by a subsequent 15 s of occlusion, which induces a blunted CRH.sub.p.sub.r.sub.e.sub.c. These results suggest that postischemic long-lasting inhibition of F.sub.0F.sub.1 **ATP synthase** activity may be a feature of the preconditioned heart. The increase in ATP concentration after **preconditioning** is in agreement with previous reports of reduced ATP hydrolysis by cytoplasmic

ATPases.

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ACCESSION NUMBER: 2004-0123853 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): ATP-sensitive K.sup.+ channels in renal **mitochondria**
AUTHOR: CANCHERINI Douglas V.; TRABUCO Leonardo G.; REBOUCAS Nancy A.; KOWALTOWSKI Alicia J.
CORPORATE SOURCE: Departamento de Fisiologia e Biofisica, Instituto de Ciencias Biomedicas, Universidade de Sao Paulo, 05508-900 Sao Paulo, Brazil; Departamento de Bioquimica, Instituto de Quimica, Universidade de Sao Paulo, 05508-900 Sao Paulo, Brazil
SOURCE: American journal of physiology. Renal physiology, (2003), 54(6), F1291-F1296, 38 refs.
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670F, 354000118779340270
TIEN ATP-sensitive K.sup.+ channels in renal **mitochondria**
AN 2004-0123853 PASCAL
CP Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
AB Isolated kidney **mitochondria** swell when incubated in hyposmotic solutions containing K.sup.+ salts in a manner inhibited by ATP, ADP, 5-hydroxydecanoate, and glibenclamide and stimulated by GTP and diazoxide. These results suggest the existence of ATP-sensitive K.sup.+ channels in these **mitochondria**, similar to those previously described in heart, liver, and brain. Renal mitochondrial ATP-sensitive K.sup.+ uptake rates are .eqvsim.140 nmol.min.sup.-.sup.1.mg protein.sup.-.sup.1. This K.sup.+ transport results in a slight increase in respiration and decrease in the inner membrane potential. In addition, the activation of ATP-inhibited K.sup.+ uptake using diazoxide leads to a decrease of ATP hydrolysis through the reverse activity of the F.sub.0F.sub.1 **ATP synthase** when respiration is inhibited. In conclusion, we characterize an ATP-sensitive K.sup.+ transport pathway in kidney **mitochondria** that affects volume, respiration, and membrane potential and may have a role in the prevention of mitochondrial ATP hydrolysis.

L4 ANSWER 8 OF 15 USPATFULL on STN

ACCESSION NUMBER: 2004:327968 USPATFULL
TITLE: Methods and compositions for modulating proteins modified in preconditioning against ischemia/hypoxia
INVENTOR(S): Eyk, Jennifer E. Van, Baltimore, MD, UNITED STATES
Elliott, Steven T., Cockeysville, MD, UNITED STATES
Arrell, David Kent, Rochester, MN, UNITED STATES

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 2004259793	A1	20041223	
APPLICATION INFO.:	US 2004-824027	A1	20040414	(10)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 2003-463139P	20030414	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C., 66 East Main Street, Marlton, NJ, 08053

NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 1334
TI Methods and compositions for modulating proteins modified in preconditioning against ischemia/hypoxia
AB Proteins modified by pharmacological preconditioning are provided. Compositions, methods and events for modulating these proteins and priming cells for preconditioning and inducing preconditioning in a cell, tissue or organ as well as methods for identifying new compositions and methods for such priming and induction are also provided. In addition, methods for diagnosing and monitoring preconditioning or ischemic, hypoxic, ischemic/reperfusion and hypoxic/reperfusion conditions or the ability of a cell, tissue or organ to survive injury by measuring modulation of one or more of these preconditioning proteins are provided.

L4 ANSWER 9 OF 15 USPATFULL on STN
ACCESSION NUMBER: 2003:176318 USPATFULL
TITLE: Methods to identify compounds affecting mitochondria
INVENTOR(S): Marban, Eduardo, Lutherville, MD, United States
O'Rourke, Brian, Sparks, MD, United States
PATENT ASSIGNEE(S): Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6586241	B1	20030701
APPLICATION INFO.:	US 2000-684730		20001006 (9)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1998-60774, filed on 15 Apr 1998, now patented, Pat. No. US 6183948
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Lankford, Jr., Leon B.
LEGAL REPRESENTATIVE: Corless, Peter F., Edwards & Angell, LLP
NUMBER OF CLAIMS: 3
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 1039
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Methods to identify compounds affecting mitochondria
AB The present invention relates to methods for identifying a compound capable of modulating mitochondrial function, comprising contacting a eukaryotic cell with one or more candidate compounds, and detecting a change in the mitochondrial redox state of the cell. The methods further relates to such methods wherein endogenous fluorescence of the cell mitochondria is indicative of a change of redox state.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 10 OF 15 USPATFULL on STN
ACCESSION NUMBER: 2003:30273 USPATFULL
TITLE: Methods and compositions for modulating adenosine triphosphate (ATP) in cells and preventing cell injury or death via post-translational modifications to **ATP synthase**
INVENTOR(S): Van Eyk, Jennifer E., Kingston, CANADA
Arrell, David Kent, Kingston, CANADA

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003022220	A1	20030130

APPLICATION INFO.: US 2002-189820 A1 20020703 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-303491P	20010706 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C., 66 E. Main Street, Marlton, NJ, 08053
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 8 Drawing Page(s)
LINE COUNT: 762
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Methods and compositions for modulating adenosine triphosphate (ATP) in cells and preventing cell injury or death via post-translational modifications to **ATP synthase**
AB Compositions and methods for modulating adenosine triphosphate (ATP) in cells via altering post-translational modifications of **ATP synthase** subunits or precursors thereof such as the **ATP synthase** β chain and its precursor are provided. These compositions and methods are useful in preconditioning organs and preventing cell injury or cell death via regulating ATP synthesis or hydrolysis in cells of the organs.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 11 OF 15 USPATFULL on STN
ACCESSION NUMBER: 2001:18196 USPATFULL
TITLE: Methods to identify compounds affecting mitochondria
INVENTOR(S): Marban, Eduardo, Lutherville, MD, United States
O'Rourke, Brian, Sparks, MD, United States
PATENT ASSIGNEE(S): Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6183948	B1	20010206
APPLICATION INFO.:	US 1998-60774		19980415 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Lankford, Jr., Leon B.
LEGAL REPRESENTATIVE: Corless, Peter F., Schray, Kerri P.Edwards & Angell, LLP
NUMBER OF CLAIMS: 31
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 6 Drawing Page(s)
LINE COUNT: 1117
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
TI Methods to identify compounds affecting mitochondria
AB The present invention relates to methods for identifying a compound capable of modulating mitochondrial function, comprising contacting a eukaryotic cell with one or more candidate compounds, and detecting a change in the mitochondrial redox state of the cell. The methods further relates to such methods wherein endogenous fluorescence of the cell mitochondria is indicative of a change of redox state.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L4 ANSWER 12 OF 15 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2004278351 ESBIOBASE
TITLE: F.sub.0F.sub.1 **ATP synthase**

activity is differently modulated by coronary reactive hyperemia before and after ischemic **preconditioning** in the goat

AUTHOR: Penna C.; Pagliaro P.; Rastaldo R.; Di Pancrazio F.; Lippe G.; Gattullo D.; Mancardi D.; Samaja M.; Losano G.; Mavelli I.

CORPORATE SOURCE: P. Pagliaro, Dipto. di Sci. Cliniche e Biologiche, Universita di Torino, Ospedale S. Luigi, Regione Gonzole, 10043 Orbassano (TO), Italy.
E-mail: pasquale.pagliaro@unito.it

SOURCE: American Journal of Physiology - Heart and Circulatory Physiology, (2004), 287/5 56-5 (H2192-H2200), 48 reference(s)
CODEN: AJPPDI ISSN: 0363-6135

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

TI F.sub.0F.sub.1 **ATP synthase** activity is differently modulated by coronary reactive hyperemia before and after ischemic **preconditioning** in the goat

AB The amplitude of coronary reactive hyperemia (CRH), elicited by 15 s of ischemia, is reduced in hearts subjected to 5 min of ischemic **preconditioning** (IP). F.sub.0F.sub.1 **ATP synthase** activity and ATP concentration are also altered by IP. We hypothesized that F.sub.0F.sub.1 **ATP synthase** is differently modulated by the inhibitor protein IF.sub.1 during CRH elicited before (CRH.sub.n.sub.p) and after (CRH.sub.p.sub.r.sub.e.sub.c) IP. Hemodynamic parameters were recorded in 10 anesthetized goats. Myocardial biopsies were obtained before IP (C.sub.n.sub.p), during CRH.sub.n.sub.p, 4 and 6 min after the onset of CRH.sub.n.sub.p, after IP (C.sub.p.sub.r.sub.e.sub.c), during CRH.sub.p.sub.r.sub.e.sub.c, and 4 min after CRH.sub.p.sub.r.sub.e.sub.c. F .sub.0F.sub.1 **ATP synthase** activity, ATP concentration, and ATP-to-ADP ratio (ATP/ADP) were determined. Compared with CRH.sub.n.sub.p, IP blunted CRH.sub.p.sub.r.sub.e.sub.c. F.sub.0F.sub.1 **ATP synthase** activity transiently increased during CRH.sub.n.sub.p, decreased 4 min after CRH .sub.n.sub.p, and returned to control 2 min later; it was lower after IP (C.sub.p.sub.r.sub.e.sub.c) and did not change during and after CRH.sub.p.sub.r.sub.e.sub.c. All these changes in activity were modulated by IF.sub.1. During CRH .sub.n.sub.p, ATP concentration and ATP/ADP were reduced compared with C .sub.n.sub.p and began to rise 6 min thereafter. During C.sub.p.sub.r.sub.e.sub.c, both parameters were transiently reduced but increased during and after CRH .sub.p.sub.r.sub.e.sub.c. Hence, during CRH.sub.n.sub.p, F.sub.0F.sub.1 **ATP synthase** activity transiently increases and then decreases significantly. The short-lasting inhibition of the enzyme may explain why a few seconds of occlusion do not induce IP. After IP, F.sub.0F.sub.1 **ATP synthase** activity is blunted, and it is not affected by a subsequent 15 s of occlusion, which induces a blunted CRH.sub.p.sub.r.sub.e.sub.c. These results suggest that postischemic long-lasting inhibition of F.sub.0F.sub.1 **ATP synthase** activity may be a feature of the preconditioned heart. The increase in ATP concentration after **preconditioning** is in agreement with previous reports of reduced ATP hydrolysis by cytoplasmic ATPases.

L4 ANSWER 13 OF 15 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2004213219 ESBIOBASE

TITLE: Multiprotein complex containing succinate dehydrogenase confers mitochondrial ATP-sensitive K.sup.+ channel activity

AUTHOR: Ardehali H.; Chen Z.; Ko Y.; Mejia-Alvarez R.; Marban E.
CORPORATE SOURCE: E. Marban, 844 Ross Building, Johns Hopkins University, 720 Rutland Avenue, Baltimore, MD 21205, United States.
E-mail: marban@jhmi.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (10 AUG 2004), 101/32 (11880-11885), 31 reference(s)
CODEN: PNASA6 ISSN: 0027-8424
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

TI Multiprotein complex containing succinate dehydrogenase confers mitochondrial ATP-sensitive K.sup.+ channel activity

AB The mitochondrial ATP-sensitive K.sup.+ (mitoK.sub.A.sub.T.sub.P) channel plays a central role in protection of cardiac and neuronal cells against ischemia and apoptosis, but its molecular structure is unknown. Succinate dehydrogenase (SDH) is inhibited by mitoK.sub.A.sub.T.sub.P activators, fueling the contrary view that SDH, rather than mitoK.sub.A.sub.T.sub.P, is the target of cardioprotective drugs. Here, we report that SDH forms part of mitoK .sub.A.sub.T.sub.P functionally and structurally. Four mitochondrial proteins [mitochondrial ATP-binding cassette protein 1 (mABC1), phosphate carrier, adenine nucleotide translocator, and **ATP synthase**] associate with SDH. A purified IM fraction containing these proteins was reconstituted into proteoliposomes and lipid bilayers and shown to confer mitoK.sub.A.sub.T.sub.P channel activity. This channel activity is sensitive not only to mitoK .sub.A.sub.T.sub.P activators and blockers but also to SDH inhibitors. These results reconcile the controversy over the basis of ischemic **preconditioning** by demonstrating that SDH is a component of mitoK.sub.A.sub.T.sub.P as part of a macromolecular super-complex. The findings also provide a tangible clue as to the structural basis of mitoK.sub.A.sub.T.sub.P channels.

L4 ANSWER 14 OF 15 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003289067 ESBIOBASE
TITLE: ATP-sensitive K.sup.+ channels in renal **mitochondria**
AUTHOR: Cancherini D.V.; Trabuco L.G.; Reboucas N.A.; Kowaltowski A.J.
CORPORATE SOURCE: A.J. Kowaltowski, Cidade Universitaria, Av. Prof. Lineu Prestes, 748, 05508-900, Sao Paulo, Brazil.
E-mail: alicia@iq.usp.br
SOURCE: American Journal of Physiology - Renal Physiology, (2003), 285/6 54-6 (F1291-F1296), 38 reference(s)
CODEN: AJPPFK ISSN: 0363-6127
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English

TI ATP-sensitive K.sup.+ channels in renal **mitochondria**

AB Isolated kidney **mitochondria** swell when incubated in hyposmotic solutions containing K.sup.+ salts in a manner inhibited by ATP, ADP, 5-hydroxydecanoate, and glibenclamide and stimulated by GTP and diazoxide. These results suggest the existence of ATP-sensitive K.sup.+ channels in these **mitochondria**, similar to those previously described in heart, liver, and brain. Renal mitochondrial ATP-sensitive K.sup.+ uptake rates are .apprx.140 nmol.midldot.min.sup.-.sup.1-mg protein.sup.-.sup.1. This K .sup.+ transport results in a slight increase in respiration and decrease in the inner membrane potential. In addition,

the activation of ATP-inhibited K.sup.+ uptake using diazoxide leads to a decrease of ATP hydrolysis through the reverse activity of the F.sub.0F.sub.1 **ATP synthase** when respiration is inhibited. In conclusion, we characterize an ATP-sensitive K.sup.+ transport pathway in kidney **mitochondria** that affects volume, respiration, and membrane potential and may have a role in the prevention of mitochondrial ATP hydrolysis.

L4 ANSWER 15 OF 15 Elsevier BIOBASE COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2002273905 ESBIOBASE
TITLE: Halothane, isoflurane and sevolfurane inhibit **NADH: Ubiquinone oxidoreductase** (complex I) of cardiac **mitochondria**
AUTHOR: Hanley P.J.; Ray J.; Brandt U.; Daut J.
CORPORATE SOURCE: J. Daut, Institute of Physiology, Marburg University, Deutschhausstrasse 2, 35037 Marburg, Germany. E-mail: daut@mailer.uni-marburg.de
SOURCE: Journal of Physiology, (01 NOV 2002), 544/3 (687-693), 40 reference(s)
CODEN: JPHYA7 ISSN: 0022-3751
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English

TI Halothane, isoflurane and sevolfurane inhibit **NADH: Ubiquinone oxidoreductase** (complex I) of cardiac **mitochondria**

AB We have investigated the effects of volatile anaesthetics on electron transport chain activity in the mammalian heart. Halothane, isoflurane and sevoflurane reversibly increased NADH fluorescence (autofluorescence) in intact ventricular myocytes of guinea-pig, suggesting that NADH oxidation was impaired. Using pig heart submitochondrial particles we found that the anaesthetics dose-dependently inhibited NADH oxidation in the order: halothane > isoflurane = sevoflurane. Succinate oxidation was unaffected by either isoflurane or sevoflurane, indicating that these agents selectively inhibit complex I (**NADH:ubiquinone oxidoreductase**). In addition to inhibiting NADH oxidation, halothane also inhibited succinate oxidation (and succinate dehydrogenase), albeit to a lesser extent. To test the hypothesis that complex I is a target of volatile anaesthetics, we examined the effects of these agents on **NADH:ubiquinone oxidoreductase** (EC 1.6.99.3) activity using the ubiquinone analogue DBQ (decylubiquinone) as substrate. Halothane, isoflurane and sevoflurane dose-dependently inhibited NADH:DBQ oxidoreductase activity. Unlike the classical inhibitor rotenone, none of the anaesthetics completely inhibited enzyme activity at high concentration, suggesting that these agents bind weakly to the 'hydrophobic inhibitory site' of complex I. In conclusion, halothane, isoflurane and sevoflurane inhibit complex I (**NADH:ubiquinone oxidoreductase**) of the electron transport chain. At concentrations of .apprx.2 MAC (minimal alveolar concentration), the activity of **NADH: ubiquinone oxidoreductase** was reduced by about 20% in the presence of halothane or isoflurane, and by about 10% in the presence of sevoflurane. These inhibitory effects are unlikely to compromise cardiac performance at usual clinical concentrations, but may contribute to the mechanism by which volatile anaesthetics induce pharmacological **preconditioning**.

=> s vaneyk,j?/au
L5 72 VANEYK,J?/AU

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=> s arrell,d?/au
L6          76 ARRELL,D?/AU

=> s elliott,s?/au
L7        3875 ELLIOTT,S?/AU

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=> s L9 AND L7 AND L8
L10          0 L9 AND L7 AND L8

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     AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,
     BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
     CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 14:23:54 ON 09 MAY 2005
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             5144   FILE PASCAL
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                3   FILE PHARMAML
               18   FILE PHIN
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                6   FILE PROUSDDR
                9   FILE RDISCLOSURE
             8243   FILE SCISEARCH
             2045   FILE TOXCENTER
             4450   FILE USPATFULL
              361   FILE USPAT2
                8   FILE VETB
               14   FILE VETU
               99   FILE WATER
              718   FILE WPIDS
                3   FILE WPIFV
              718   FILE WPINDEX
L1               QUE PRECONDITIONING
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     FILE 'SCISEARCH, BIOSIS, CAPLUS, PASCAL, MEDLINE, USPATFULL, EMBASE,
     ESBIOBASE, TOXCENTER' ENTERED AT 14:25:03 ON 09 MAY 2005
L2          1261 S PRECONDITIONING (P) MITOCHONDRIA
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L4            15 S L3 AND (ISOCITRATE(W)DEHYDROGENASE(W)NAD(W)ALPHA(W)SUBUNIT O
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L6            76 S ARRELL,D?/AU
L7          3875 S ELLIOTT,S?/AU
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L13  ANSWER 1 OF 2     MEDLINE on STN
TI   [Molecular modifications of troponin I and T detected in serum from
     patients with acute myocardial infarction].
     Molekylaere forandringer af troponin I og T pavist i serum hos patienter
     med akut myokardieinfarkt.

L13  ANSWER 2 OF 2  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation  on STN
TI   Identification of myofilament proteins that are degraded and/or released
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during ischemia/reperfusion: Effects on the pCa-force relation.

=> d l13 ibib ti abs 1-2

L13 ANSWER 1 OF 2 MEDLINE on STN
ACCESSION NUMBER: 2003044477 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12553091
TITLE: [Molecular modifications of troponin I and T detected in serum from patients with acute myocardial infarction]. Molekylaere forandringer af troponin I og T pavist i serum hos patienter med akut myokardieinfarkt.
AUTHOR: Atar Dan; Madsen Lene Helleskov; Labugger Ralf; **VanEyk Jennifer E**
CORPORATE SOURCE: H:S Frederiksberg Hospital, kardiologisk klinik E, DK-2000 Frederiksberg.. datar@dadlnet.dk
SOURCE: Ugeskrift for laeger, (2003 Jan 6) 165 (2) 116-20. Journal code: 0141730. ISSN: 0041-5782.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Danish
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20030130
Last Updated on STN: 20030228
Entered Medline: 20030227

TI [Molecular modifications of troponin I and T detected in serum from patients with acute myocardial infarction]. Molekylaere forandringer af troponin I og T pavist i serum hos patienter med akut myokardieinfarkt.

AB INTRODUCTION: Cardiac troponin I and T (cTnI and cTnT) are specific biochemical serum markers for acute myocardial infarction (AMI). However, cTnI diagnostic assays are plagued by difficulties, resulting in > 20-fold differences in measured values. These discrepancies may result from the release of the numerous cTnI modification products that are present in ischaemic myocardium. The resolution of these discrepancies requires an investigation of the exact forms of the troponins present in the bloodstream of patients after myocardial injury. MATERIAL AND METHODS: A Westernblot direct serum analysis protocol was developed that allowed us to detect intact cTnI and a spectrum of up to 11 modified products in the serum from patients with AMI. RESULTS: We document both a cTnI degradation pattern and the existence of phosphorylated cTnI in serum. The number and extent of these modifications reflect patterns similar to the time profiles of the routine clinical serum markers of total creatine kinase, creatine kinase-MB, and cTnI (determined by ELISA). Data from in vitro experiments, which were undertaken to study the degradation of human recombinant cTnI and cTnT when spiked in serum, indicate that some modification products present in patient serum existed in the myocardium. DISCUSSION: This pilot study defines, for the first time, what forms of cTnI and cTnT appear in the bloodstream of AMI patients, and it clarifies the lack of standardization between different cTnI diagnostic assays.

L13 ANSWER 2 OF 2 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1997:4179 BIOSIS
DOCUMENT NUMBER: PREV199799303382
TITLE: Identification of myofilament proteins that are degraded and/or released during ischemia/reperfusion: Effects on the pCa-force relation.
AUTHOR(S): **Vaneyk, Jennifer E.** [Reprint author]; Powers, Francis M.; Law, William R.; Hodges, Robert S.; Solaro, R. John
CORPORATE SOURCE: Univ. Ill., Chicago, IL, USA
SOURCE: Circulation, (1996) Vol. 94, No. 8 SUPPL., pp. I365.

Meeting Info.: 69th Scientific Sessions of the American Heart Association. New Orleans, Louisiana, USA. November 10-13, 1996.
CODEN: CIRCAZ. ISSN: 0009-7322.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Jan 1997
Last Updated on STN: 7 Jan 1997
TI Identification of myofilament proteins that are degraded and/or released during ischemia/reperfusion: Effects on the pCa-force relation.

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(FILE 'HOME' ENTERED AT 14:23:43 ON 09 MAY 2005)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 14:23:54 ON 09 MAY 2005

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4	FILE IMSRESEARCH

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               14   FILE VETU
               99   FILE WATER
              718   FILE WPIDS
                3   FILE WPIFV
              718   FILE WPINDEX
L1               QUE PRECONDITIONING
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     FILE 'SCISEARCH, BIOSIS, CAPLUS, PASCAL, MEDLINE, USPATFULL, EMBASE,
     ESBIOBASE, TOXCENTER' ENTERED AT 14:25:03 ON 09 MAY 2005
L2          1261 S PRECONDITIONING (P) MITOCHONDRIA
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L4            15 S L3 AND (ISOCITRATE(W)DEHYDROGENASE(W)NAD(W)ALPHA(W)SUBUNIT O
L5            72 S VANEYK,J?/AU
L6            76 S ARRELL,D?/AU
L7          3875 S ELLIOTT,S?/AU
L8             0 S L5 AND L6 AND L7
L9             6 S EYK,J?/AU
L10            0 S L9 AND L7 AND L8
L11            0 S VANEYK,JENNIFER/AU
L12            2 S VANEYK,JENNIFER?/AU
L13            2 DUP REM L12 (0 DUPLICATES REMOVED)

=> s L6 AND L7
L14            5 L6 AND L7

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15            5 DUP REM L14 (0 DUPLICATES REMOVED)

=> d l15 ibib ti abs 1-5

L15  ANSWER 1 OF 5  USPATFULL on STN
ACCESSION NUMBER:         2004:327968 USPATFULL
TITLE:                    Methods and compositions for modulating proteins
                          modified in preconditioning against ischemia/hypoxia
INVENTOR(S):              Eyk, Jennifer E. Van, Baltimore, MD, UNITED STATES
                            Elliott, Steven T., Cockeysville, MD, UNITED
                          STATES
                            Arrell, David Kent, Rochester, MN, UNITED
                          STATES

                               NUMBER       KIND    DATE
```

PATENT INFORMATION:	US 2004259793	A1	20041223
APPLICATION INFO.:	US 2004-824027	A1	20040414 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-463139P	20030414 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Licata & Tyrrell P.C., 66 East Main Street, Marlton, NJ, 08053
NUMBER OF CLAIMS: 12
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 12 Drawing Page(s)
LINE COUNT: 1334

TI Methods and compositions for modulating proteins modified in preconditioning against ischemia/hypoxia

AB Proteins modified by pharmacological preconditioning are provided. Compositions, methods and events for modulating these proteins and priming cells for preconditioning and inducing preconditioning in a cell, tissue or organ as well as methods for identifying new compositions and methods for such priming and induction are also provided. In addition, methods for diagnosing and monitoring preconditioning or ischemic, hypoxic, ischemic/reperfusion and hypoxic/reperfusion conditions or the ability of a cell, tissue or organ to survive injury by measuring modulation of one or more of these preconditioning proteins are provided.

L15 ANSWER 2 OF 5 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:40526 BIOSIS
DOCUMENT NUMBER: PREV200400041897
TITLE: 2-Dimensional gel electrophoresis proteomic database of rabbit ventricular myocytes.
AUTHOR(S): **Elliott, S.** [Reprint Author]; **Arrell, D. Kent** [Reprint Author]; Doherty-Kirby, A.; Brown, H. [Reprint Author]; Lajoie, G.; Marban, E.; Van Eyk, J. [Reprint Author]
CORPORATE SOURCE: Queen's University, Kingston, ON, Canada
SOURCE: Molecular & Cellular Proteomics, (September 2003) Vol. 2, No. 9, pp. 835. print.
Meeting Info.: HUPO (Human Proteomics Organisation) 2nd Annual and IUBMB (International Union of Biochemistry and Molecular Biology) XIX World Congress. Montreal, Quebec, Canada. October 08-11, 2003. American Society for Biochemistry and Molecular Biology Inc.
ISSN: 1535-9476 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jan 2004
Last Updated on STN: 14 Jan 2004

TI 2-Dimensional gel electrophoresis proteomic database of rabbit ventricular myocytes.

L15 ANSWER 3 OF 5 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:162755 SCISEARCH
THE GENUINE ARTICLE: 511EY
TITLE: Preconditioning post-translationally modifies cardiac mitochondrial F1Fo ATPase subunit
AUTHOR: **Arrell D K (Reprint)**; Neverova I; **Elliott S T**; Turcotte A; Van Eyk J E

CORPORATE SOURCE: Queens Univ, Kingston, ON K7L 3N6, Canada
COUNTRY OF AUTHOR: Canada
SOURCE: BIOPHYSICAL JOURNAL, (JAN 2002) Vol. 82, No. 1, Part 2, pp. 611A-611A. MA 2986.
Publisher: BIOPHYSICAL SOCIETY, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA.
ISSN: 0006-3495.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
TI Preconditioning post-translationally modifies cardiac mitochondrial F1Fo ATPase subunit

L15 ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:365381 BIOSIS
DOCUMENT NUMBER: PREV200200365381
TITLE: Preconditioning post-translationally modifies cardiac mitochondrial F1Fo ATPase beta subunit.
AUTHOR(S): **Arrell, D. Kent** [Reprint author]; Neverova, Irina [Reprint author]; **Elliott, Steven T.** [Reprint author]; Turcotte, Antony [Reprint author]; Van Eyk, Jennifer E. [Reprint author]
CORPORATE SOURCE: Queen's University, 414 Botterell Hall, Kingston, Ontario, K7L 3N6, Canada
SOURCE: Biophysical Journal, (January, 2002) Vol. 82, No. 1 Part 2, pp. 611a. print.
Meeting Info.: 46th Annual Meeting of the Biophysical Society. San Francisco, California, USA. February 23-27, 2002.
CODEN: BIOJAU. ISSN: 0006-3495.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jul 2002
Last Updated on STN: 3 Jul 2002
TI Preconditioning post-translationally modifies cardiac mitochondrial F1Fo ATPase beta subunit.

L15 ANSWER 5 OF 5 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:687876 SCISEARCH
THE GENUINE ARTICLE: 579YR
TITLE: Proteomic analysis of preconditioning reveals post-translational modification of the F1F0 ATPase beta subunit
AUTHOR: **Arrell D K (Reprint); Elliott S;** Neverova I; Marban E; Van Eyk J E
CORPORATE SOURCE: Queens Univ, Dept Physiol, Kingston, ON, Canada; Queens Univ, Dept Biochem, Kingston, ON, Canada; Johns Hopkins Univ, Inst Mol Cardiobiol, Baltimore, MD USA
COUNTRY OF AUTHOR: Canada; USA
SOURCE: JOURNAL OF MOLECULAR AND CELLULAR CARDIOLOGY, (JUL 2002) Vol. 34, No. 7, pp. A33-A33.
Publisher: ACADEMIC PRESS LTD ELSEVIER SCIENCE LTD, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.
ISSN: 0022-2828.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0
TI Proteomic analysis of preconditioning reveals post-translational modification of the F1F0 ATPase beta subunit

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=> FIL STNGUIDE
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                        132.69     134.08

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)              SINCE FILE      TOTAL
                                                           ENTRY    SESSION
CA SUBSCRIBER PRICE                                         -1.46      -1.46

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AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: May  6, 2005 (20050506/UP).

=> d his

     (FILE 'HOME' ENTERED AT 14:23:43 ON 09 MAY 2005)

     INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE,
     AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS,
     BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB,
     CROPU, DDFB, DDFU, DGENE, DISSABS, ...' ENTERED AT 14:23:54 ON 09 MAY 2005
               SEA PRECONDITIONING
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             370   FILE ADISCTI
              18   FILE ADISINSIGHT
              14   FILE ADISNEWS
             275   FILE AGRICOLA
              70   FILE ANABSTR
              14   FILE ANTE
              27   FILE AQUALINE
             140   FILE AQUASCI
             121   FILE BIOBUSINESS
               4   FILE BIOCOMMERCE
             131   FILE BIOENG
            6800   FILE BIOSIS
             109   FILE BIOTECHABS
             109   FILE BIOTECHDS
             412   FILE BIOTECHNO
             657   FILE CABA
             315   FILE CANCERLIT
            5254   FILE CAPLUS
              54   FILE CEABA-VTB
              15   FILE CIN
             267   FILE CONFSCI
              15   FILE CROPB
              59   FILE CROPU
               5   FILE DDFB
             869   FILE DDFU
              76   FILE DGENE
             553   FILE DISSABS
               5   FILE DRUGB
             994   FILE DRUGU
              82   FILE EMBAL
            4241   FILE EMBASE
            2600   FILE ESBIOBASE
             206   FILE FEDRIP
              65   FILE FROSTI
              98   FILE FSTA
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              61   FILE GENBANK
               9   FILE HEALSAFE
            1201   FILE IFIPAT
               3   FILE IMSDRUGNEWS
               4   FILE IMSRESEARCH
             711   FILE JICST-EPLUS
               7   FILE KOSMET
             526   FILE LIFESCI
               6   FILE MEDICONF
            4881   FILE MEDLINE
              24   FILE NIOSHTIC
             583   FILE NTIS
              75   FILE OCEAN
            5144   FILE PASCAL
               2   FILE PHAR
               3   FILE PHARMAML
              18   FILE PHIN
             306   FILE PROMT
               6   FILE PROUSDDR
               9   FILE RDISCLOSURE
            8243   FILE SCISEARCH
            2045   FILE TOXCENTER
            4450   FILE USPATFULL
             361   FILE USPAT2
               8   FILE VETB
              14   FILE VETU
              99   FILE WATER
             718   FILE WPIDS
               3   FILE WPIFV
             718   FILE WPINDEX
L1              QUE PRECONDITIONING
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     FILE 'SCISEARCH, BIOSIS, CAPLUS, PASCAL, MEDLINE, USPATFULL, EMBASE,
     ESBIOBASE, TOXCENTER' ENTERED AT 14:25:03 ON 09 MAY 2005
L2          1261 S PRECONDITIONING (P) MITOCHONDRIA
L3           508 DUP REM L2 (753 DUPLICATES REMOVED)
L4            15 S L3 AND (ISOCITRATE(W)DEHYDROGENASE(W)NAD(W)ALPHA(W)SUBUNIT O
L5            72 S VANEYK,J?/AU
L6            76 S ARRELL,D?/AU
L7          3875 S ELLIOTT,S?/AU
L8             0 S L5 AND L6 AND L7
L9             6 S EYK,J?/AU
L10            0 S L9 AND L7 AND L8
L11            0 S VANEYK,JENNIFER/AU
L12            2 S VANEYK,JENNIFER?/AU
L13            2 DUP REM L12 (0 DUPLICATES REMOVED)
L14            5 S L6 AND L7
L15            5 DUP REM L14 (0 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 14:38:33 ON 09 MAY 2005

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---Logging off of STN---


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Executing the logoff script...

=> LOG Y
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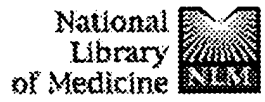
COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.54	134.62

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-1.46

STN INTERNATIONAL LOGOFF AT 14:43:40 ON 09 MAY 2005





All Databases PubMed Nucleotide Protein Genome Structure OMIM PMC Jou

Search PubMed for #1 AND (preconditioning) Preview Go Cle

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- To combine searches use # before search number, e.g., #2 AND #6.
- Search numbers may not be continuous; all searches are represented.
- Click on query # to add to strategy

Search	Most Recent Queries	Time	Result
#27	Search **#1 AND (preconditioning)**	10:41:42	0
#21	Search **(isocitrate dehydrogenase NAD alpha subunit OR succinyl CoA ligase OR NADH ubiquinone oxidoreductase OR ATP synthase) AND (preconditioning)**	10:34:51	51
#18	Search **#7 OR DJ-1 AND (preconditioning)**	10:08:34	88
#17	Search **#7 OR DJ-1**	09:53:30	10586
#11	Search **DJ-1**	09:51:38	107
#8	Search **RNA binding protein regulatory subunit DJ-1**	09:36:22	2
#7	Search **isocitrate dehydrogenase NAD alpha subunit OR succinyl CoA ligase OR NADH ubiquinone oxidoreductase OR ATP synthase OR prohibitin OR ADP ribosyl hydrolase OR HSP27**	09:35:52	10484
#6	Search **isocitrate dehydrogenase NAD alpha subunit OR succinyl CoA ligase OR NADH ubiquinone oxidoreductase**	09:30:35	3138
#5	Search **level of isocitrate dehydrogenase NAD OR succinyl CoA ligase OR NADH ubiquinone oxidoreductase**	09:29:15	3281
#4	Search **level of isocitrate dehydrogenase NAD alpha subunit**	09:27:11	1
#2	Search **level of isocitrate dehydrogenase NAD AND alpha subunit**	09:24:45	1
#1	Search **level of isocitrate dehydrogenase NAD**	09:24:22	159

Clear History